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Dwek Institute for Cancer Therapy Research

Department of Biological Regulation and Dwek Institute for Cancer Therapy Research

CANCER RESEARCH CLUB

The role of the ubiquitin system in Ras-driven diseases

Prof. Anna Sablina

Head of the Lab for Mechanisms of Cell Transformation

VIB Center for Cancer Biology

Department of Oncology, University of Leuven
Belgium

19 December Thursday 2019

14:00

Candiotty Auditorium

The superfamily of Ras GTPases emerges as a central regulatory node for coordinating cell signaling. About one-third of human cancers have somatic mutations in one of the three RAS genes. The RAS pathway is also hyperactivated in a significant subset of human pathologies that lack activating mutations of RAS, suggesting that RAS signaling may be frequently activated, independently of genetic alterations in RAS genes, by alternative mechanisms not yet fully elucidated.

During our recent studies, we have partially uncovered the molecular machinery controlling reversible ubiquitination of the RAS protein network. Strikingly, loss of function of positive regulators of RAS ubiquitination is associated with a wide range of human disease, whereas a negative regulator of RAS ubiquitination, OTUB1, is commonly amplified and overexpressed in wild-type RAS epithelial cancers. Thus, the genetic evidence and our initial functional data explicitly indicate that aberrations in the ubiquitin system are implicated in the pathogenesis of RAS driven disease. Understanding unconventional mechanisms of RAS activation will not only allow us to identify patients who might benefit from RAS pathway inhibitors, but will likely lead toward novel therapeutic approaches for these patients.

Host

Prof. Rony Seger
Department of Biological Regulation

Faculty of Biology

For more information and assistance with accessibility issues, please contact

Yam Nof ✉ yam.nof@weizmann.ac.il

Melanie Koloditzky ✉ melaniek@weizmann.ac.il